(FILE 'HOME' ENTERED AT 13:28:10 ON 22 NOV 2006)

FILE 'BIOSIS, MEDLINE, HCAPLUS, CABA, JAPIO, AGRICOLA, PASCAL, USPATFULL, SCISEARCH, CONFSCI, EPFULL' ENTERED AT 13:28:27 ON 22 NOV 2006

```
L1       298991 S (PYLORI OR HP OR HPYLORI OR HELICOBACER OR CAMPYLOBACTER)
L2       303501 S (PYLORI OR HP OR HPYLORI OR HELICOBACTER OR CAMPYLOBACTER)
L3        46075 S  (PEPSINOGEN I OR PEPSINOGEN-I OR PEPSINOGEN A OR PG-I OR PGA
L4         2262 S L2 AND L3
L5           96 S L4 AND (H,K-ATPASE OR ATPASE)
L6           74 S L5 AND ANTIBOD?
L7            1 S L6 AND MULTIPLY
L8           40 S L6 AND GASTRITIS
L9           19 S L8 AND DIAGNOSIS
L10           9 DUP REM L9 (10 DUPLICATES REMOVED)
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FILE 'STNGUIDE' ENTERED AT 13:36:52 ON 22 NOV 2006

=>

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=> FIL
BIOSIS,MEDLINE,HCAPLUS,CABA,JAPIO,AGRICOLA,PASCAL,USPATFULL,SCISEARCH,CONFSCI,EPFULL
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'BIOSIS' ENTERED AT 13:28:27 ON 22 NOV 2006
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FILE 'MEDLINE' ENTERED AT 13:28:27 ON 22 NOV 2006

FILE 'HCAPLUS' ENTERED AT 13:28:27 ON 22 NOV 2006
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FILE 'JAPIO' ENTERED AT 13:28:27 ON 22 NOV 2006
COPYRIGHT (C) 2006 Japanese Patent Office (JPO)- JAPIO

FILE 'AGRICOLA' ENTERED AT 13:28:27 ON 22 NOV 2006

FILE 'PASCAL' ENTERED AT 13:28:27 ON 22 NOV 2006
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FILE 'USPATFULL' ENTERED AT 13:28:27 ON 22 NOV 2006
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FILE 'SCISEARCH' ENTERED AT 13:28:27 ON 22 NOV 2006
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FILE 'CONFSCI' ENTERED AT 13:28:27 ON 22 NOV 2006
COPYRIGHT (C) 2006 Cambridge Scientific Abstracts (CSA)

FILE 'EPFULL' ENTERED AT 13:28:27 ON 22 NOV 2006
COPYRIGHT (C) 2006 European Patent Office / FIZ Karlsruhe

=> s (pylori or hp or hpylori or helicobacer or campylobacter)
L1      298991 (PYLORI OR HP OR HPYLORI OR HELICOBACER OR CAMPYLOBACTER)

=> s (pylori or hp or hpylori or helicobacter or campylobacter)
L2      303501 (PYLORI OR HP OR HPYLORI OR HELICOBACTER OR CAMPYLOBACTER)

=> s  (pepsinogen I or pepsinogen-I or pepsinogen A or PG-I or pga or pgi)
   7 FILES SEARCHED...
L3      46075 (PEPSINOGEN I OR PEPSINOGEN-I OR PEPSINOGEN A OR PG-I OR PGA OR
              PGI)

=> s l2 and l3
L4        2262 L2 AND L3

=> s l4 and (H,K-ATPase or ATPase)
L5          96 L4 AND (H,K-ATPASE OR ATPASE)

=> s L5 and antibod?
L6          74 L5 AND ANTIBOD?
```

=> s l6 and multiply
L7 1 L6 AND MULTIPLY

=> d l7 ibib abs

L7 ANSWER 1 OF 1 USPATFULL on STN

ACCESSION NUMBER: 2005:177322 USPATFULL

TITLE: Corynebacterium glutamicum genes encoding regulatory proteins

INVENTOR(S): Pompejus, Markus, Waldsee, GERMANY, FEDERAL REPUBLIC OF
Kroger, Burkhard, Limburgerhof, GERMANY, FEDERAL REPUBLIC OF
Schroder, Hartwig, Nubloch, GERMANY, FEDERAL REPUBLIC OF
Zelder, Oskar, Speyer, GERMANY, FEDERAL REPUBLIC OF
Haberhauer, Gregor, Limburgerhof, GERMANY, FEDERAL REPUBLIC OF

PATENT ASSIGNEE(S): BASF AG, Ludwigshafen, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005153402	A1	20050714
APPLICATION INFO.:	US 2004-6098	A1	20041206 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-602874, filed on 23 Jun 2000, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1999-19930476	19990701
	DE 1999-19931419	19990708
	DE 1999-19931420	19990708
	DE 1999-19932122	19990709
	DE 1999-19932128	19990709
	DE 1999-19932134	19990709
	DE 1999-19932206	19990709
	DE 1999-19932207	19990709
	DE 1999-19933003	19990714
	DE 1999-19941390	19990831
	DE 1999-19942088	19990903
	DE 1999-19942124	19990903
	US 1999-141031P	19990625 (60)
	US 1999-142690P	19990701 (60)
	US 1999-151251P	19990827 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LAHIVE & COCKFIELD, LLP., 28 STATE STREET, BOSTON, MA, 02109, US
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
LINE COUNT: 6287
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Isolated nucleic acid molecules, designated MR nucleic acid molecules, which encode novel MR proteins from Corynebacterium glutamicum are described. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing MR nucleic acid molecules, and host cells into which the expression vectors have been introduced. The invention still further provides isolated MR proteins, mutated MR proteins, fusion proteins, antigenic peptides and methods for the improvement of production of a desired compound from C. glutamicum based on genetic engineering of MR genes in this organism.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

```
=> s l6 and gastritis
L8          40 L6 AND GASTRITIS

=> s l8 and diagnosis
L9          19 L8 AND DIAGNOSIS

=> rem dup l9
DUP IS NOT VALID HERE
The DELETE command is used to remove various items stored by the
system.

To delete a saved query, saved answer set, saved L-number list, SDI
request, batch request, mailing list, or user-defined cluster, format,
or search field, enter the name.  The name may include ?  for left,
right, or simultaneous left and right truncation.

Examples:

  DELETE BIO?/Q           - delete query names starting with BIO
  DELETE ?DRUG/A          - delete answer set names ending with DRUG
  DELETE ?ELEC?/L         - delete L-number lists containing ELEC
  DELETE ANTICOAG/S       - delete SDI request
  DELETE ENZYME/B         - delete batch request
  DELETE .MYCLUSTER       - delete user-defined cluster
  DELETE .MYFORMAT        - delete user-defined display format
  DELETE .MYFIELD         - delete user-defined search field
  DELETE NAMELIST MYLIST - delete mailing list

To delete an ordered document or an offline print, enter its
number.

Examples:

  DELETE P123001C         - delete print request
  DELETE D134002C         - delete document order request

To delete an individual L-number or range of L-numbers, enter the
L-number or L-number range.  You may also enter DELETE LAST followed
by a number, n, to delete the last n L-numbers.  RENUMBER or
NORENUMBER may also be explicitly specified to override the value of
SET RENUMBER.

Examples:

  DELETE L21              - delete a single L-number
  DELETE L3-L6            - delete a range of L-numbers
  DELETE LAST 4           - delete the last 4 L-numbers
  DELETE L33-             - delete L33 and any higher L-number
  DELETE -L55             - delete L55 and any lower L-number
  DELETE L2-L6 RENUMBER - delete a range of L-numbers and
                          renumber remaining L-numbers
  DELETE RENUMBER         - renumber L-numbers after deletion of
                          intermediate L-numbers

Entire sets of saved items, SDI requests, batch requests, user-defined
items, or E-numbers can be deleted.

Examples:

  DELETE SAVED/Q - delete all saved queries
  DELETE SAVED/A - delete all saved answer sets
```

```
  DELETE SAVED/L - delete all saved L-number lists
  DELETE SAVED   - delete all saved queries, answer sets,
                   and L-number lists
  DELETE SAVED/S - delete all SDI requests
  DELETE SAVED/B - delete all batch requests
  DELETE CLUSTER - delete all user-defined clusters
  DELETE FORMAT  - delete all user-defined display formats
  DELETE FIELD   - delete all user-defined search fields
  DELETE SELECT  - delete all E-numbers
  DELETE HISTORY - delete all L-numbers and restart the
                   session at L1

To delete an entire multifile SDI request, enter DELETE and
the name of the request.  To delete a component from the
multifile SDI, enter DELETE and the name of the component.

=> dup rem 19
PROCESSING COMPLETED FOR L9
L10            9 DUP REM L9 (10 DUPLICATES REMOVED)

=> d 19 1-9 abs ibib

L9   ANSWER 1 OF 19  BIOSIS  COPYRIGHT (c) 2006 The Thomson Corporation  on STN
AB   Juvenile patients affected with autoimmune thyroid disorders showed a
     14-21% prevalence of parietal cell antibodies (PCA) reacting
     against the H+/K+-ATPase of the gastric
     parietal cells.  PCA are the principal immunological markers of atrophic
     body gastritis (ABG).  ABG is characterized by loss of oxyntic
     glands, achlorhydria, and hypergastrinemia.  The aim of this study was to
     determine whether PCA positivity could be associated with biochemical and
     histological manifestations of gastric autoimmunity in juvenile patients
     with autoimmune thyroid disease (AITD).  We studied 129 children ( 96
     females and 33 males) with chronic lymphocytic thyroiditis (n = 115) or
     Graves' disease (n = 14).  Mean age at diagnosis of AITD was 9.7
     +/- 3.3 yr, and mean age at sampling was 12.3 +/- 3.7 yr.  We determined
     PCA and Helicobacter pylori antibodies,
     gastrin, and pepsinogen I plasma levels.  Gastroscopy
     with multiple biopsies was carried out in a subgroup of patients with PCA
     positivity.  We found that 30% of children had detectable PCA.
     Hypergastrinemia was found in 45% of the PCA-positive children (range,
     40-675 pg/ml) vs. 12% of PCA-negative children (range, 35-65 pg/ml; P
     0.001).  Eighteen patients with PCA positivity underwent gastroscopy;
     eight of these children had normogastrinemia, which showed no signs of
     ABG, and 10 children had hypergastrinemia, of whom five had mild to severe
     ABG.  Our study shows that autoimmune gastritis is an early
     event in juvenile AITD with detectable PCA.  Gastrin plasma level is a
     reliable marker of gastric atrophy.
ACCESSION NUMBER:    2005:22114  BIOSIS
DOCUMENT NUMBER:     PREV200500021412
TITLE:               Early manifestations of gastric autoimmunity in patients
                     with juvenile autoimmune thyroid diseases.
AUTHOR(S):           Segni, Maria [Reprint Author]; Borrelli, Osvaldo;
                     Pucarelli, Ida; Delle Fave, Gianfranco; Pasquino, Anna
                     Maria; Annibale, Bruno
CORPORATE SOURCE:    Dept PediatSch Med 1, Univ La Sapienza, Via Giuseppe
                     Vaccari 3, I-00194, Rome, Italy
                     m.segni@mclink.it
SOURCE:              Journal of Clinical Endocrinology & Metabolism, (October
                     2004) Vol. 89, No. 10, pp. 4944-4948. print.
                     ISSN: 0021-972X (ISSN print).
DOCUMENT TYPE:       Article
LANGUAGE:            English
ENTRY DATE:          Entered STN: 29 Dec 2004
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Last Updated on STN: 29 Dec 2004

L9 ANSWER 2 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AB Background: Gastroscopy and examination of biopsy is normally required for diagnosis of gastritis. This is costly and inconvenient for the patient, and there is a need for a simple pregastroscopic screening method to reduce the endoscopy workload. Our aim was to develop a serological screening test for gastritis. Methods: Sera from subjects examined with gastroscopy and biopsy were analyzed for H ,K-ATPase antibodies, Helicobacter pylori antibodies and pepsinogen I. The diagnoses were normal gastric mucosa (n=50), duodenal ulcer (n=53) and atrophic corpus gastritis, with (n=50) or without pernicious anemia (n=46). Results: An evaluation scheme was constructed to optimize the diagnostic agreement between serology and gastric mucosal morphology. The sensitivity to detect gastritis was 98% (146/149) (95% CI 94-100%) and the specificity 84% (42/50) (95% CI 71-93%). Additional sera from 483 subjects from the general population were analyzed. There was a good agreement between serology and gastric mucosal morphology. Conclusions: Assays of multiple serum analytes are useful for the initial screening of gastritis. They are complementary to upper gastroscopy by identification of subjects with a normal gastric mucosa, those who qualify for eradication of H. pylori, and those who have developed atrophy and are at risk of developing malignancy and, therefore, require gastroscopic examination.

ACCESSION NUMBER: 2002:389474 BIOSIS
DOCUMENT NUMBER: PREV200200389474
TITLE: Diagnosis of gastritis by means of a combination of serological analyses.
AUTHOR(S): Mardh, Erik; Mardh, Sven [Reprint author]; Mardh, Bibbi; Borch, Kurt
CORPORATE SOURCE: Department of Biomedicine and Surgery, Faculty of Health Sciences, Linkoping University, S-581 85, Linkoping, Sweden sven.mardh@mcb.liu.se
SOURCE: Clinica Chimica Acta, (June, 2002) Vol. 320, No. 1-2, pp. 17-27. print.
CODEN: CCATAR. ISSN: 0009-8981.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Jul 2002
Last Updated on STN: 17 Jul 2002

L9 ANSWER 3 OF 19 MEDLINE on STN
AB Juvenile patients affected with autoimmune thyroid disorders showed a 14-21% prevalence of parietal cell antibodies (PCA) reacting against the H+/K+-ATPase of the gastric parietal cells. PCA are the principal immunological markers of atrophic body gastritis (ABG).ABG is characterized by loss of oxyntic glands, achlorhydria, and hypergastrinemia. The aim of this study was to determine whether PCA positivity could be associated with biochemical and histological manifestations of gastric autoimmunity in juvenile patients with autoimmune thyroid disease (AITD). We studied 129 children (96 females and 33 males) with chronic lymphocytic thyroiditis (n = 115) or Graves' disease (n = 14). Mean age at diagnosis of AITD was 9.7 +/- 3.3 yr, and mean age at sampling was 12.3 +/- 3.7 yr.We determined PCA and Helicobacter pylori antibodies, gastrin, and pepsinogen I plasma levels. Gastroscopy with multiple biopsies was carried out in a subgroup of patients with PCA positivity. We found that 30% of children had detectable PCA. Hypergastrinemia was found in 45% of the PCA-positive children (range, 40-675 pg/ml) vs. 12% of PCA-negative children (range, 35-65 pg/ml; P < 0.001). Eighteen patients with PCA positivity underwent gastroscopy; eight of these children had normogastrinemia, which showed no signs of

ABG, and 10 children had hypergastrinemia, of whom five had mild to severe ABG. Our study shows that autoimmune gastritis is an early event in juvenile AITD with detectable PCA. Gastrin plasma level is a reliable marker of gastric atrophy.

ACCESSION NUMBER: 2004503383 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15472189
TITLE: Early manifestations of gastric autoimmunity in patients with juvenile autoimmune thyroid diseases.
AUTHOR: Segni Maria; Borrelli Osvaldo; Pucarelli Ida; Delle Fave Gianfranco; Pasquino Anna Maria; Annibale Bruno
CORPORATE SOURCE: Department of Pediatrics, II Medical School, University La Sapienza, I-00161 Rome, Italy. m.segni@mclink.it. <m.segni@mclink.it>
SOURCE: The Journal of clinical endocrinology and metabolism, (2004 Oct) Vol. 89, No. 10, pp. 4944-8.
Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200411
ENTRY DATE: Entered STN: 9 Oct 2004
Last Updated on STN: 19 Dec 2004
Entered Medline: 19 Nov 2004

L9 ANSWER 4 OF 19 MEDLINE on STN

AB BACKGROUND: Gastroscopy and examination of biopsy is normally required for diagnosis of gastritis. This is costly and inconvenient for the patient, and there is a need for a simple pregastroscopic screening method to reduce the endoscopy workload. Our aim was to develop a serological screening test for gastritis. METHODS: Sera from subjects examined with gastroscopy and biopsy were analyzed for H ,K-ATPase antibodies, Helicobacter pylori antibodies and pepsinogen I. The diagnoses were normal gastric mucosa (n=50), duodenal ulcer (n=53) and atrophic corpus gastritis, with (n=50) or without pernicious anemia (n=46). RESULTS: An evaluation scheme was constructed to optimize the diagnostic agreement between serology and gastric mucosal morphology. The sensitivity to detect gastritis was 98% (146/149) (95% CI 94-100%) and the specificity 84% (42/50) (95% CI 71-93%). Additional sera from 483 subjects from the general population were analyzed. There was a good agreement between serology and gastric mucosal morphology. CONCLUSIONS: Assays of multiple serum analytes are useful for the initial screening of gastritis. They are complementary to upper gastroscopy by identification of subjects with a normal gastric mucosa, those who qualify for eradication of H. pylori, and those who have developed atrophy and are at risk of developing malignancy and, therefore, require gastroscopic examination.

ACCESSION NUMBER: 2002244937 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11983196
TITLE: Diagnosis of gastritis by means of a combination of serological analyses.
AUTHOR: Mardh Erik; Mardh Sven; Mardh Bibbi; Borch Kurt
CORPORATE SOURCE: Department of Biomedicine and Surgery, Faculty of Health Sciences, Linkoping University, S-581 85 Linkoping, Sweden.
SOURCE: Clinica chimica acta; international journal of clinical chemistry, (2002 Jun) Vol. 320, No. 1-2, pp. 17-27.
Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207

ENTRY DATE: Entered STN: 2 May 2002
Last Updated on STN: 25 Jul 2002
Entered Medline: 24 Jul 2002

L9 ANSWER 5 OF 19 MEDLINE on STN
AB OBJECTIVES: To compare the diagnostic performance of serum antibodies to H+,K+-ATPase (EC 3.6.1.36), serum pepsinogen A (EC 3.4.23.1) and the Schilling test in diagnosing chronic atrophic body gastritis; to study the interrelationships between H+,K+-ATPase antibodies, serology for Helicobacter pylori, and gastric morphology. DESIGN: Patients with suspected cobalamin deficiency and serum cobalamin < 200 micromol/l were investigated using upper gastrointestinal endoscopy, the Schilling test and serum tests for H+,K+-ATPase antibodies, pepsinogen A, and H. pylori. SETTING: The Department of Internal Medicine, Sahlgrenska University Hospital, Goteborg, Sweden. PATIENTS: Ninety seven consecutively referred patients. MAIN OUTCOME MEASURES: Sensitivity and specificity of assays for serum H+,K+-ATPase antibodies, serum pepsinogen A, and the Schilling test. RESULTS: Assays of serum antibodies to H+,K+-ATPase and of serum pepsinogen A displayed equal diagnostic sensitivity for atrophic gastritis (around 0.90 for the severe forms) and higher than that for the Schilling test (0.65). The diagnostic specificity for pepsinogen A (1.0) was higher than for H+, K+-ATPase antibodies (about 0.80). The prevalence of antral gastritis and positivity for H. pylori antibodies declined with the transition of body gastritis into severe atrophy, while the prevalence of H+,K+-ATPase antibodies increased. CONCLUSION: Pepsinogen A is preferable to serum H+,K+-ATPase antibodies in the diagnosis of gastric body mucosal atrophy. The formation of H+,K+-ATPase antibodies does not seem to be a primary event in the development of gastric body muscosal atrophy.
ACCESSION NUMBER: 1999070725 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9855083
TITLE: Serum antibodies to H+,K+-ATPase, serum pepsinogen A and Helicobacter pylori in relation to gastric mucosa morphology in patients with low or low-normal concentrations of serum cobalamins.
AUTHOR: Lindgren A; Burman P; Kilander A F; Nilsson O; Lindstedt G
CORPORATE SOURCE: Department of Internal Medicine, Sahlgrenska University Hospital, Goteborg, Sweden.
SOURCE: European journal of gastroenterology & hepatology, (1998 Jul) Vol. 10, No. 7, pp. 583-8.
Journal code: 9000874. ISSN: 0954-691X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 23 Feb 1999
Last Updated on STN: 23 Feb 1999
Entered Medline: 11 Feb 1999

L9 ANSWER 6 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN
AB Juvenile patients affected with autoimmune thyroid disorders showed a 14-21% prevalence of parietal cell antibodies (PCA) reacting

against the H+/K+-ATPase of the gastric parietal cells. PCA are the principal immunol. markers of atrophic body gastritis (ABG). ABG is characterized by loss of oxyntic glands, achlorhydria, and hypergastrinemia. The aim of this study was to determine whether PCA positivity could be associated with biochem. and histol. manifestations of gastric autoimmunity in juvenile patients with autoimmune thyroid disease (AITD). We studied 129 children (96 females and 33 males) with chronic lymphocytic thyroiditis (n = 115) or Graves' disease (n = 14). Mean age at diagnosis of AITD was 9.7 ± 3.3 yr, and mean age at sampling was 12.3 ± 3.7 yr. We determined PCA and Helicobacter pylori antibodies, gastrin, and pepsinogen I plasma levels. Gastroscopy with multiple biopsies was carried out in a subgroup of patients with PCA positivity. We found that 30% of children had detectable PCA. Hypergastrinemia was found in 45% of the PCA-pos. children (range, 40-675 pg/mL) vs. 12% of PCA-neg. children (range, 35-65 pg/mL; P < 0.001). Eighteen patients with PCA positivity underwent gastroscopy; eight of these children had normogastrinemia, which showed no signs of ABG, and 10 children had hypergastrinemia, of whom five had mild to severe ABG. Our study shows that autoimmune gastritis is an early event in juvenile AITD with detectable PCA. Gastrin plasma level is a reliable marker of gastric atrophy.

ACCESSION NUMBER: 2004:883503 HCAPLUS
DOCUMENT NUMBER: 142:5289
TITLE: Early manifestations of gastric autoimmunity in patients with juvenile autoimmune thyroid diseases
AUTHOR(S): Segni, Maria; Borrelli, Osvaldo; Pucarelli, Ida; Delle Fave, Gianfranco; Pasquino, Anna Maria; Annibale, Bruno
CORPORATE SOURCE: Department of Pediatrics, II Medical School, University "La Sapienza,", Rome, I-00161, Italy
SOURCE: Journal of Clinical Endocrinology and Metabolism (2004), 89(10), 4944-4948
CODEN: JCEMAZ; ISSN: 0021-972X
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN
AB The present invention relates to a method for diagnosing possible presence of gastritis in a human by evaluating a blood sample, comprising assaying the blood sample for the presence of antibodies specific for H,K-ATPase, antibodies specific for Helicobacter pylori, and the concentration of pepsinogen I, whereby the presence of H, K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen I concentration are compared between themselves and in relation to the resp. values of H,K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen concentration of a normal population, in a software related system, wherein altered levels in the sample is indicative of gastritis, and whereby, preferably, an altered level detection leads to the issuance of a remittance for further investigation with regard to gastritis. ELISAs were performed on blood samples.

ACCESSION NUMBER: 2003:778075 HCAPLUS
DOCUMENT NUMBER: 139:273239
TITLE: Screening method and kit for gastritis by determining pepsinogen I, H,K-ATPase

antibodies, and Helicobacter pylori antibodies in blood

INVENTOR(S): Mardh, Sven; Mardh, Erik
PATENT ASSIGNEE(S): Atrophus AB, Swed.
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003081248	A1	20031002	WO 2003-SE469	20030321
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2478891	AA	20031002	CA 2003-2478891	20030321
AU 2003216007	A1	20031008	AU 2003-216007	20030321
EP 1488238	A1	20041222	EP 2003-745056	20030321
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005521055	T2	20050714	JP 2003-578931	20030321
CN 1701235	A	20051123	CN 2003-806799	20030321
NZ 535937	A	20060831	NZ 2003-535937	20030321

PRIORITY APPLN. INFO.: SE 2002-974 A 20020327
WO 2003-SE469 W 20030321

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN

AB Background: Gastroscopy and examination of biopsy is normally required for diagnosis of gastritis. This is costly and inconvenient for the patient, and there is a need for a simple pregastroscopic screening method to reduce the endoscopy workload. Our aim was to develop a serol. screening test for gastritis. Methods: Sera from subjects examined with gastroscopy and biopsy were analyzed for H, K-ATPase antibodies, Helicobacter pylori antibodies and pepsinogen I. The diagnoses were normal gastric mucosa (n=50), duodenal ulcer (n=53) and atrophic corpus gastritis, with (n=50) or without pernicious anemia (n=46). Results: An evaluation scheme was constructed to optimize the diagnostic agreement between serol. and gastric mucosal morphol. The sensitivity to detect gastritis was 98% (146/149) (95% CI 94-100%) and the specificity 84% (42/50) (95% CI 71-93%). Addnl. sera from 483 subjects from the general population were analyzed. There was a good agreement between serol. and gastric mucosal morphol. Conclusions: Assays of multiple serum analytes are useful for the initial screening of gastritis. They are complementary to upper gastroscopy by identification of subjects with a normal gastric mucosa, those who qualify for eradication of H. pylori, and those who have developed atrophy and are at risk of developing malignancy and, therefore, require gastroscopic examination

ACCESSION NUMBER: 2002:320397 HCAPLUS
TITLE: Diagnosis of gastritis by means of a combination of serological analyses

AUTHOR(S): Mardh, Erik; Mardh, Sven; Mardh, Bibbi; Borch, Kurt
CORPORATE SOURCE: Department of Biomedicine and Surgery, Faculty of Health Sciences, Linkoping University, Linkoping, S-581 85, Swed.
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CODEN: CCATAR; ISSN: 0009-8981
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DOCUMENT TYPE: Journal
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REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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AN 2004-0574817 PASCAL
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AB Juvenile patients affected with autoimmune thyroid disorders showed a 14-21% prevalence of parietal cell antibodies (PCA) reacting against the H+/K+-ATPase of the gastric parietal cells. PCA are the principal immunological markers of atrophic body gastritis (ABG). ABG is characterized by loss of oxyntic glands, achlorhydria, and hypergastrinemia. The aim of this study was to determine whether PCA positivity could be associated with biochemical and histological manifestations of gastric autoimmunity in juvenile patients with autoimmune thyroid disease (AITD). We studied 129 children (96 females and 33 males) with chronic lymphocytic thyroiditis (n = 115) or Graves' disease (n = 14). Mean age at diagnosis of AITD was 9.7 ± 3.3 yr, and mean age at sampling was 12.3 ± 3.7 yr. We determined PCA and Helicobacter pylori antibodies, gastrin, and pepsinogen I plasma levels. Gastroscopy with multiple biopsies was carried out in a subgroup of patients with PCA positivity. We found that 30% of children had detectable PCA. Hypergastrinemia was found in 45% of the PCA-positive children (range, 40-675 pg/ml) vs. 12% of PCA-negative children (range, 35-65 pg/ml; $P < 0.001$). Eighteen patients with PCA positivity underwent gastroscopy; eight of these children had normogastrinemia, which showed no signs of ABG, and 10 children had hypergastrinemia, of whom five had mild to severe ABG. Our study shows that autoimmune gastritis is an early event in juvenile AITD with detectable PCA. Gastrin plasma level is a reliable marker of gastric atrophy.
ACCESSION NUMBER: 2004-0574817 PASCAL
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TITLE (IN ENGLISH): Early manifestations of gastric autoimmunity in patients with juvenile autoimmune thyroid diseases
AUTHOR: SEGNI Maria; BORRELLI Osvaldo; PUCARELLI Ida; DELLE FAVE Gianfranco; PASQUINO Anna Maria; ANNIBALE Bruno
CORPORATE SOURCE: Department of Pediatrics, I Medical School, University "La Sapienza", 00161 Rome, Italy; Department of Gastroenterology, II Medical School, University "La Sapienza", 00161 Rome, Italy
SOURCE: The Journal of clinical endocrinology and metabolism, (2004), 89(10), 4944-4948, 33 refs.
ISSN: 0021-972X CODEN: JCEMAZ
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☐ 2. 5567594. 20 Dec 93; 22 Oct 96. Methods and compositions for the detection and treatment of diseases associated with antigens of microorganisms. Calenoff; Emanuel. 435/7.32; 435/7.33 435/822 435/883 435/975 436/513 436/518. G01N033/53 G01N033/554 G01N033/569 .

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L15: Entry 1 of 2 File: USPT Sep 21, 2004

US-PAT-NO: 6794153
DOCUMENT-IDENTIFIER: US 6794153 B2

TITLE: Helicobacter pylori antigens in blood

DATE-ISSUED: September 21, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
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Hung; Chung-Ho	Burlingame	CA		

US-CL-CURRENT: 435/7.32; 424/184.1, 435/7.1, 435/7.2, 435/7.35, 435/7.37, 435/7.92, 435/7.93, 435/7.94, 435/975, 436/518 , 436/526, 436/806, 436/824

CLAIMS:

What is claimed is:

1. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent; removing said dissociation reagent from said serum to form a dissociation reagent-removed serum sample; providing a first antibody against Helicobacter pylori, wherein said first antibody is a lyclonal antibody purified by an affinity column with potassium thiocynate (KSCN) in water; contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori, wherein said second antibody is a polyclonal antibody purified by an affinity column with potassium thiocynate (KSCN) in water; wherein said first antibody and said second antibody are eluted from said affinity column with potassium thiocynate (KSCN) in water; wherein one of said first and second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of said Helicobacter pylori antigen by measuring the presence of the detection agent in said second complex.

2. The method according to claim 1, wherein said solid carrier is polyethylene, polystyrene, polypropylene, or a nitrocellulose membrane.

3. The method according to claim 1, wherein said detection agent comprises at least one selected from the group consisting of an enzymatic marker, a fluorescent agent, a luminescent agent, a radioactive label, and a color particle.

4. The method according to claim 3, wherein said enzymatic marker comprises an alkaline phosphatase or horseradish peroxidase.

5. The method according to claim 3, wherein said fluorescent or luminescent agent comprises at least one selected from the group consisting of fluorescein, rhodamine, europium, luminol, and acridium.

6. The method according to claim 3, wherein said color particle comprises at least one consisting of gold, silver, blue latex, and selenium.

7. The method according to claim 3, wherein said first antibody is bound to said solid carrier and said second antibody is labelled with said enzymatic marker.

8. The method according to claim 3, wherein said first antibody is labelled with said color particle and said second antibody is bound to said solid carrier.

9. The method according to claim 1, wherein said dissociation reagent is removed from said sample by dilution, filtration, column chromatography, or dialysis.

10. The method according to claim 1, further comprising treating said dissociation reagent-removed serum sample with a protein based reagent.

11. The method according to claim 10, wherein said protein based reagent is at least one selected from the group consisting of fetal bovine serum, pig serum, goat serum, horse serum, casein, albumin, gelatin, and bovine serum albumin.

12. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent; wherein said dissociation reagent comprises a NaCl or KCl solution at a concentration between 0.2 M to 1.5 M; removing said dissociation reagent; providing a first antibody against Helicobactur pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column; contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori, wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein one of said first and second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of said Helicobacter pylori antigen by measuring the presence of the detection agent in said second complex.

13. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent; wherein said dissociation reagent comprises a detergent which comprises at least one selected from the group consisting of sodium dodecyl sulphate (SDS), TWEEN 20, octylglucoside deoxycholate, and TRITON X-100 at a concentration between 0.1 to 2.0% by weight; removing said dissociation reagent: providing a first antibody against Helicobacter pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column: contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein one of said first and second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of said Helicobacter pylori antigen by measuring the presence of the detection agent in said second complex.

14. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent; wherein said dissociation reagent comprises an organic solvent which comprises at least one selected from the group consisting of dioxane and ethylene glycol; removing said dissociation reagent; providing a first antibody against Helicobacter pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column; contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein one of said first end second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of said Helicobacter pylori, antigen by measuring the presence of the detection agent in said second complex.

15. The method according to claim 14, wherein said dioxane is at a concentration of about 10% by weight and ethylene glycol is at a concentration of 40% by weight.

16. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a

human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent; wherein said dissociation reagent comprises a chaotropic agent which is selected from the group consisting of guanidine HCl, urea, and potassium thiocyanate (KSCN); removing said dissociation reagent; providing a first antibody against Helicobacter pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column; contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori, wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein one of said first and second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of said Helicobacter pylori antigen by measuring the presence of the detection agent in said second complex.

17. The method according to claim 16, wherein said guanidine HCl is at a molarity of 0.5 to 6 M, said urea is at molarity of 0.5 to 8 M, and said KSCN is at a molarity of 0.5to 3 M.

18. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms or peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent; wherein said dissociation reagent comprises at least one enzyme which is selected from the group consisting of protease and lipase; removing said dissociation reagent; providing a first antibody against Helicobacter pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column; contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori, wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein one of said first and second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of said Helicobacter pylori antigen by measuring the presence of the detection agent in said second complex.

19. The method according to claim 18, wherein said protease is at least one selected from the group consisting of trypsin, chymotrypsin, pepsin, V8 protease, and subtilisin.

20. The method according to claim 18, wherein said protease is at a concentration of 1 to 10 units per ml of serum.

21. The method according to claim 18, wherein said lipase is either a lipoprotein lipase from bovine milk or lipase from Candida rugosa.

22. The method according to claim 21, wherein said lipase is at a concentration of 1 to 10 units per ml of serum.

23. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample with a dissociation reagent, wherein said dissociation reagent is (a) a NaCl or KCl solution at a concentration between 0.2 M to 1.5 M, (b) a detergent which is sodium dodecyl sulphate (SDS), TWEEN 20, octylgluoside, deoxycholate, or TRITON X-100 at a concentration between 0.1 to 2.0% by weight, (c) an organic solvent which is either dioxane or ethylene glycol, (d) a chaotropic agent which is guanidine HCl, urea, or potassium thiocyanate (KSCN), (e) an enzyme which is a protease or a lipase, or (f) any combination of (a)-(e); removing said dissociation reagent; providing a first antibody against Helicobacter pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column; contacting said dissociation reagent-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori; wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein said first antibody and said second antibody are eluted from said affinity column; wherein said first antibody is bound to a solid carrier; contacting said first complex with the second antibody to form a second complex; preparing a secondary antibody against an antibody-producing animal species for said second antibody; labeling said secondary antibody with a detection agent to form a detection agent-labeled secondary antibody; contacting said

second complex with said detection agent-labeled secondary antibody to form a third complex; and detecting the presence of said Helicobacter pylori antigen by measuring the presence of the detection agent in said third complex.

24. The method according to claim 23, wherein said a detection agent comprises at least one selected from the group consisting of an enzymatic marker, a fluorescent agent, a luminescent agent, a radioactive label, and a color particle.

25. A method for detecting a Helicobacter pylori antigen in a serum sample obtained from a human comprising: providing said serum sample from said human; wherein said human has symptoms of peptic ulcer disease and/or chronic gastritis; treating said serum sample at a dissociation condition to form a dissociation condition-treated serum sample; wherein said dissociation condition is obtained by changing pH of said serum sample to alkaline or acidic pH or by elevating temperature of said serum sample; wherein said sample dissociation condition comprises elevating said serum sample to a temperature of no less than 50.degree. C.; and wherein upon completion of said dissociation condition, said serum pH or said elevated serum temperature is returned to original condition; providing a first antibody against Helicobacter pylori, wherein said first antibody is a polyclonal antibody purified by an affinity column; contacting said dissociation condition-treated serum sample with the first antibody to form a first complex; providing a second antibody against Helicobacter pylori, wherein said second antibody is a polyclonal antibody purified by an affinity column; wherein one of said first and second antibody is bound to a solid carrier, the other is labeled with a detection agent; contacting said first complex with the second antibody to form a second complex; and detecting the presence of Helicobacter pylori in said second complex by measuring the presence of the detection agent in said second complex.

End of Result Set

L15: Entry 2 of 2 File: USPT Oct 22, 1996

US-PAT-NO: 5567594
DOCUMENT-IDENTIFIER: US 5567594 A
**** See image for Certificate of Correction ****

TITLE: Methods and compositions for the detection and treatment of diseases associated with antigens of microorganisms

DATE-ISSUED: October 22, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Calenoff; Emanuel	Chicago	IL		

US-CL-CURRENT: 435/7.32; 435/7.33, 435/822, 435/883, 435/975, 436/513, 436/518

CLAIMS:

What is claimed is:

1. A method of detecting in an individual a condition associated with a microorganism, said microorganism selected from the group consisting of a bacterium, a virus, and a mycoplasma, said method comprising:

a. obtaining a library of purified and isolated antigens from a single species of microorganism wherein said library is specific for the condition;

b. measuring an antigen/antibody reaction between (i) immunoglobulin E in a biological sample from the individual, wherein immunoglobulin E is the antibody, and (ii) the isolated antigens of a library of purified antigens specific for the microorganism, and

c. determining whether the condition is present, wherein the presence of said antigen/antibody reaction for said antigens indicates the presence of said condition in the individual.

2. The method of claim 1, wherein the microorganism is a bacterium.

3. The method of claim 2, wherein the condition is peptic ulcer disease and the bacterium is Helicobacter pylori.

4. The method of claim 2, wherein the condition is gastritis and the bacterium is Helicobacter pylori.

5. The method of claim 2, wherein the condition is gastric cancer and the bacterium is Helicobacter pylori.

6. The method of claim 2, wherein the condition is nasal polyposis and the bacterium is Staphyloccus aureus.

7. The method of claim 2, wherein the condition is hyperplastic sinusitis and the bacterium is Staphylococcus aureus.

8. The method of claim 1, wherein the antigens are isolated by a process comprising the steps of (i) solubilizing an extract of said microorganism in increasing concentrations of

SDS to form fractions and (ii) subfractionating each solubilized fraction by precipitation in increasing concentrations of acetone; and (iii) selecting isolated antigens having a homogeneous molecular weight that are observed as single bands on SDS-PAGE.

9. A method of measuring in a biological sample immunoglobulin E which complexes with a bacterial antigen library, the method comprising:

(a) reacting the sample with a bacterial antigenic library coupled to a solid support, wherein said library consists of isolated and purified antigens from the same bacterial species, and wherein each antigen in the library has a homogeneous molecular weight;

(b) washing and reacting the support with labelled anti-immunoglobulin E; and

(c) detecting the labeled anti-immunoglobulin E bound to the solid support.

10. A method of determining whether an individual has an immunological response to a library of bacterial antigens, said library comprising purified and isolated antigens each having a homogenous molecular weight the method comprising:

(a) providing a biological sample from an individual suspected of containing immunoglobulin E directed to antigens of the library;

(b) providing a composition consisting essentially of isolated antigens of the library;

(c) reacting the biological sample of (a) with the composition of (b) under conditions that allow immunological binding between immunoglobulin E and an antigen to which it is directed; and

(d) detecting complexes formed, if any, between immunoglobulin E in the serum of (a) and a protein antigen in the composition of (b) such that the presence of the immunoglobulin is inferred and whether an individual has an immunological response is determined.

11. The method of claim 10, wherein the serum provided has been reacted with a composition capable of removing IgA and IgG from the serum in amount sufficient to remove IgG and IgA which interferes with formation of the IgE-allergen complex.

12. The method of claim 10 wherein the composition of Step b comprises protein allergens of H. pylori.

13. A diagnostic kit comprising a library of isolated and purified microbial antigens from the same microorganism each antigen in solution in a suitable container, and means for detecting immunological complexes formed between the library of antigens from the same microorganism and immunoglobulin E in a biological sample.

14. The kit of claim 13, wherein the library is specific for H. pylori.

15. The kit of claim 13, wherein the antigens are coupled to a solid support.

End of Result Set

L11: Entry 1 of 1 File: USPT Oct 17, 2006

US-PAT-NO: 7122320
DOCUMENT-IDENTIFIER: US 7122320 B2

TITLE: Method for detecting acid-resistant microorganisms in the stool

DATE-ISSUED: October 17, 2006

PRIOR-PUBLICATION:

DOC-ID	DATE
US 20040023316 A1	February 5, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Reiter; Christian	Karlsfeld			DE
Cullman; Gerhard	Munich			DE
Friedrichs; Ulrike	Leipzig			DE
Heppner; Petra	Pullach			DE
Lakner; Meret	Munich			DE
Ringeis; Achim	Grafelfing			DE

US-CL-CURRENT: 435/7.1; 435/7.2, 435/7.32, 435/7.92, 435/7.93, 435/7.94, 435/7.95

ABSTRACT:

The invention relates to a method for detecting an infection of a mammal with an acid-resistant microorganism, wherein (a) a stool sample of a mammal is incubated with at least two different monoclonal antibodies, fragments or derivatives thereof or aptamers under conditions allowing a complex formation of antigens of the acid-resistant microorganism with antibodies, fragments or derivatives thereof or the aptamers, and wherein (aa) the first monoclonal antibody or the fragment or the derivative thereof or the first aptamer specifically binds an epitope of the first antigen, which shows at least with some mammals a structure after the intestinal passage that corresponds to the native structure or the structure which a mammal produces antibodies against after being infected or immunised with the acid-resistant microorganism or an extract or lysate thereof or a protein therefrom or a fragment thereof or a synthetic peptide; (ab) the second monoclonal antibody or the fragment or the derivative thereof or the second aptamer specifically binds an epitope of a second antigen differing from the epitope of the first antigen, which shows at least with some mammals a structure after the intestinal passage that corresponds to the native structure or the structure which a mammal produces antibodies against after being infected or immunised with the acid-resistant microorganism or an extract or lysate thereof or a protein therefrom or a fragment thereof or a synthetic peptide, wherein the parts of the mammals may overlap according to (aa) and (ab) and in total essentially make up the overall number of infected mammals; and (b) the formation of at least one antigen-antibody complex or antigen-aptamer complex according to (aa) or (ab) is detected.

46 Claims, 8 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 8

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1: Borch K, Skarsgard J, Franzen L, Mardh S, Rehfeld JF. Related Articles, Links

Benign gastric polyps: morphological and functional origin.
Dig Dis Sci. 2003 Jul;48(7):1292-7.
PMID: 12870785 [PubMed - indexed for MEDLINE]

2: Sundbom M, Mardh E, Mardh S, Ohrvall M, Gustavsson S. Related Articles, Links

Reduction in serum pepsinogen I after Roux-en-Y gastric bypass.
J Gastrointest Surg. 2003 May-Jun;7(4):529-35.
PMID: 12763411 [PubMed - indexed for MEDLINE]

3: Mardh E, Mardh S, Mardh B, Borch K. Related Articles, Links

Diagnosis of gastritis by means of a combination of serological analyses.
Clin Chim Acta. 2002 Jun;320(1-2):17-27.
PMID: 11983196 [PubMed - indexed for MEDLINE]

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2: Mardh E, Mardh S, Mardh B, Borch K. Related Articles, Links

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Clin Chim Acta. 2002 Jun;320(1-2):17-27.
PMID: 11983196 [PubMed - indexed for MEDLINE]

1: Clin Chim Acta. 2002 Jun;320(1-2):17-27.

Diagnosis of gastritis by means of a combination of serological analyses.

Mardh E, **Mardh S**, **Mardh B**, **Borch K**.

Department of Biomedicine and Surgery, Faculty of Health Sciences, Linkoping University, S-581 85 Linkoping, Sweden.

BACKGROUND: Gastroscopy and examination of biopsy is normally required for diagnosis of gastritis. This is costly and inconvenient for the patient, and there is a need for a simple pregastroscopic screening method to reduce the endoscopy workload. Our aim was to develop a serological screening test for gastritis. METHODS: Sera from subjects examined with gastroscopy and biopsy were analyzed for H,K-ATPase antibodies, Helicobacter pylori antibodies and pepsinogen I. The diagnoses were normal gastric mucosa (n=50), duodenal ulcer (n=53) and atrophic corpus gastritis, with (n=50) or without pernicious anemia (n=46). RESULTS: An evaluation scheme was constructed to optimize the diagnostic agreement between serology and gastric mucosal morphology. The sensitivity to detect gastritis was 98% (146/149) (95% CI 94-100%) and the specificity 84% (42/50) (95% CI 71-93%). Additional sera from 483 subjects from the general population were analyzed. There was a good agreement between serology and gastric mucosal morphology. CONCLUSIONS: Assays of multiple serum analytes are useful for the initial screening of gastritis. They are complementary to upper gastroscopy by identification of subjects with a normal gastric mucosa, those who qualify for eradication of H. pylori, and those who have developed atrophy and are at risk of developing malignancy and, therefore, require gastroscopic examination.

PMID: 11983196 [PubMed - indexed for MEDLINE]

negative test for current H. pylori infection: a report on histology and H. pylori detection in the gastric remnant. [Scand J Gastroenterol. 2005]

Serum levels of amidated gastrin-17 and pepsinogen I in atrophic gastritis: an observational case-control study. [Scand J Gastroenterol. 2002]

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